ACTA SCIENTIFIC NEUROLOGY (ISSN: 2582-1121)

Volume 7 Issue 5 May 2024

Editorial

Study of Hemochromosis and its Cure

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Received: March 21, 2024

Published: April 01, 2024

Abstract

Hemochromosis are inherited disorders resulting in mutation of gene involved in regulating iron metabolism. Studies of HFEC282Y.

Homozygousity this genotype was associated with elevation of serum and transferrin saturation and with iron stores more than 4 grams in females.

Keywords: Genetics; Genome; Homozygous; Hemochromosis; HFEC282Y; Mutation

Introduction

Condition (gene)	Chromosomal location	Inheritance pattern	Population affected	Relative frequency	Mechanism
HFE-hemochromatosis (HFE)	6p21	Autosomal recessive	Caucasion	Common	Decreased hepcidin production
Transferrin receptor 2 hemochromatosis (TfR2)	7q22	Autosomal recessive	Italian, ?others	Rare	Decreased hepcidin production
Juvenile hemochromatosis (HJV)	1q21	Autosomal recessive	Caucasian, others	Rare	Decreased hepcidin production
Juvenile hemochromatosis (HAMP)	19q13	Autosomal recessive	Caucasian, others	Rare	Decreased hepcidin production
Ferroportin disease (SLC40A1)	2q32	Autosomal dominant	Caucasian, others	?	Resistance to hep-cidin

Table 1: Hereditary iron overload in which anemia is present [1].

Dietary Iron absorption

Iron in the diet is present as either heme iron or non-heme iron. Most dietary non-heme iron is in the form of Fe3+, which must first be reduced to Fe2+ before it can be [2] transported across the brush border membrane by DMT1. This reduction step is likely catalysed by the brush border reductase, although other reductases may also be [3] involved. Once inside the enterocyte, the newly absorbed iron enters the intracellular iron pool. If the iron is not required by the

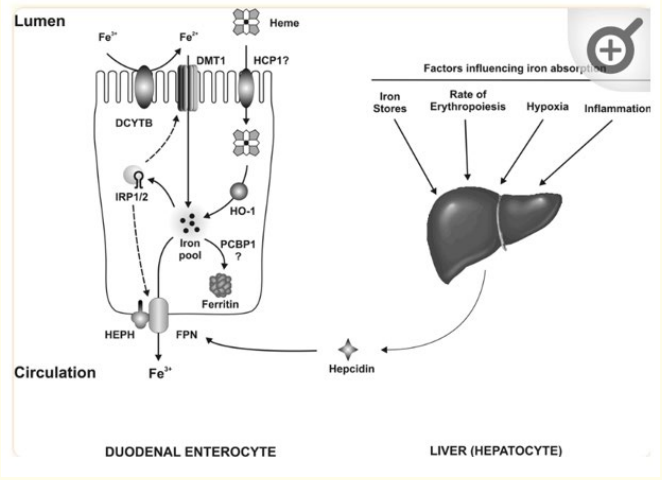

Figure 1

body it is loaded onto the iron storage protein [4] ferritin, a process possibly mediated by the iron chaperone PCBP1. Iron [5] required by the body is transferred across the basolateral membrane by FPN. The export [6] of iron also requires the ferroxidase hephaestin (HEPH), although the precise role of this protein is not known. The uptake of heme iron by enterocytes is not as well [7] understood. HCP1 can transport heme; however, its principal role appears to be the uptake of folate and its role in heme absorption remains unclear [8]. Once heme has been transported into the enterocytes the iron is released from the porphyrin ring by heme oxygenise 1 (HO-1), after which it enters the intracellular iron pool [9]. Iron absorption is regulated both by systemic signals and by local iron levels. Systemic factors influencing body iron requirements are detected in the liver and affect the expression of hepcidin, which binds to FPN and induces its internalization and degradation, thereby reducing absorption. Local iron concentrations alter IRP RNA-binding activity, which in turn may affect the levels of DMT1 and FPN. These changes serve to maintain enterocyte iron levels within defined limits despite changes in dietary iron intake [10].

Treatment

Genetherapy by inserting the normal gene HFEC282Y in monoclonal antibodies will correct disorder.

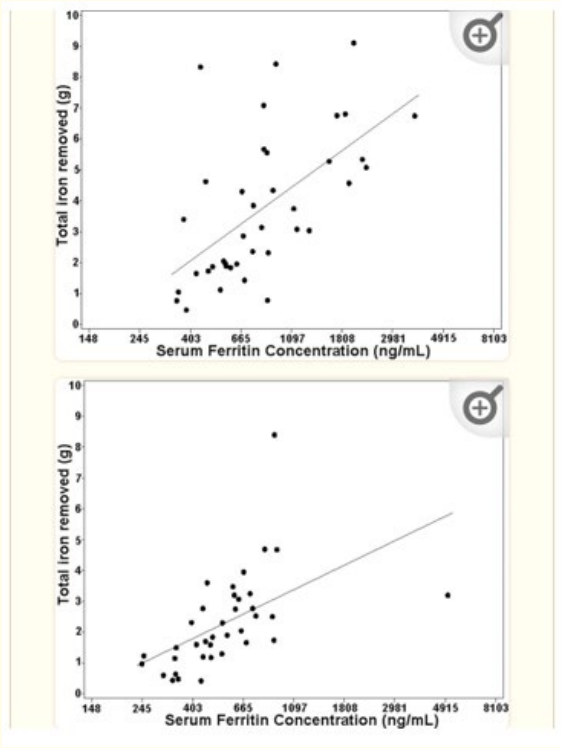

Figure 2: It gives the report of total iron clearance and serum fertillin plot graphs in women who covered botomy program plot is shown clearly below.

Stemcell therapy by replacing bonemarrow cells with correct HFEC282Y will cure the diseases and restore normal metabolism of iron (in younger age in infant stage).

Discussion

- Hemochromatosis mechanism
- Iron metabolism affect during hemochromatosis
- Cure of hemochromatosis

Conclusion

Cure of hemochromatosis was found.

Conflict of Interest

Author declare their is no conflict of interest.

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